SHORT REPORT

Rapid Progressive Graft Calcification After Aortobiiliac Bypass

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Introduction: We report an extremely rare case of rapid progressive calcification in the vascular prosthesis after aortobiiliac bypass surgery.
Report: A 56-year-old man presented to our institution with bilateral intermittent claudication. We performed aortobiiliac bypass with a gelatin-coated woven bifurcated graft. One and a half years later, the patient complained of recurrence of intermittent claudication, and abdominal computed tomography scan showed a localized severely calcified stenosis in the right leg of the vascular prosthesis.
Discussion: Few reports have been published on when, where, and how graft occlusion occurs. Physicians and surgeons should be aware of the possibility of such a condition after bypass procedures.

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INTRODUCTION

Whereas thrombotic occlusion of the implanted vascular prosthesis sometimes happens, calcified stenosis is an extremely rare condition. We report a case of rapid progressive calcification in the vascular prosthesis after aortobiiliac bypass surgery.

REPORT

A 56-year-old man presented to our institution with a 3-year history of bilateral intermittent claudication. His symptoms had recently worsened and he complained of shortened walking distances (<200 m). He had well-controlled hypertension and dyslipidemia on medication, but had no history of aortitis, atrial fibrillation, or hemodialysis.

The ankle–brachial pressure index (ABI) showed significant lower limb ischemia (right: 0.45, left: 0.49). Computed tomography showed calcified complete occlusion of the distal abdominal aorta (Fig. 1A).

We performed aortobiiliac bypass with a gelatin-coated woven bifurcated graft (J-Graft SHIELD NEO, 14 × 7 mm; Japan Lifeline Co., Ltd., Tokyo, Japan). The graft was anastomosed in an end-to-side fashion to the aorta proximally, and to bilateral common iliac arteries distally. The patient's ABI (right: 0.89, left: 1.00) and intermittent claudication were remarkably improved after surgery.

One and a half years later (6 months after the last evaluation, Fig. 1B), the patient complained of recurrence of intermittent claudication, particularly in the right lower extremity. His ABI had slightly worsened (right: 0.83, left: 0.98), and an abdominal computed tomography scan showed a localized severely calcified stenosis in the right leg of the vascular prosthesis (Fig. 1C and D). He underwent percutaneous stent implantation (Express LD, 8 × 27 mm; Boston Scientific, MA, USA) after intravascular ultrasound (Fig. 1E) with an acceptable luminal gain and no residual gradient (40 to 0 mmHg). He was discharged asymptomatic 2 days later.

DISCUSSION

Several reports have been published regarding bifurcated graft patency for aortoiliac occlusive disease, and its long-term patency rate appears to be satisfactory.[1,2] However, few reports have been published on when, where, and how graft occlusion occurs. To the best of our knowledge, there are no reports of such rapid progressive (in 6 months), localized (far from the anastomosis site), calcified stenosis of the implanted vascular prosthesis. From a technical point of view, this was not a perfect operation because of kinking of the distal anastomosis. Acute thrombotic occlusion or gradually worsening intimal thickness may occur at the site of kinking; however, we think the kinking would not lead to such rapid progressive calcified occlusion far from the anastomosis.

Although our patient had hypertension and dyslipidemia, he was generally a healthy middle-aged man. His current status was well-controlled with an antihypertensive agent and statin, as well as continued aspirin administration after initial surgery. There was no evidence of hormonal abnormalities in calcium metabolism (Ca: 9.5 mg/dL, P: 3.8 mg/dL, parathyroid hormone-intact: 28 pg/mL). The etiology of this condition is still unclear, and physicians and surgeons should be aware of the possibility of such a condition after bypass procedures.

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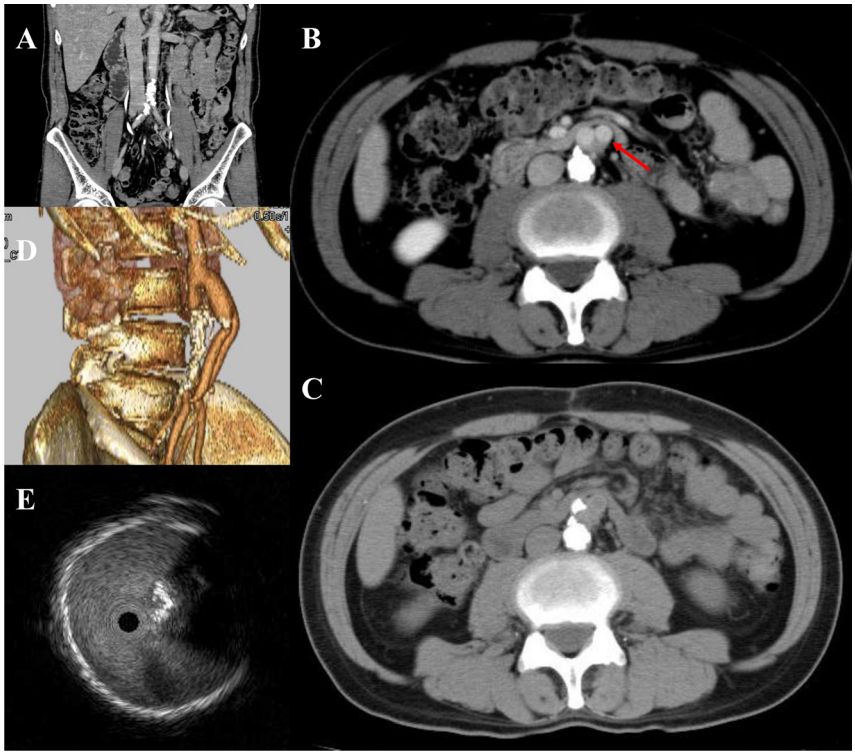

Figure 1. (A) A coronal view of preoperative computed tomography shows severe calcified stenosis of the distal abdominal aorta. (B) Computed tomography (1 year after the initial operation) shows no evidence of calcification in the implanted vascular prosthesis (red arrow). (C) Computed tomography (1.5 years after the initial operation) shows localized calcified stenosis in the right leg of the vascular prosthesis. (D) Three-dimensional computed tomography shows that the calcified lesion is located far from the anastomosis site. (E) Intravascular ultrasound finding of the calcified site.

CONFLICT OF INTEREST

None.

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REFERENCES

1 Lee GC, Yang SS, Park KM, Park Y, Kim YW, Park KB, et al. Ten year outcomes after bypass surgery in aortoiliac occlusive disease. *J Korean Surg Soc* 2012;**82**:365—9.
2 Brewster DC. Current controversies in the management of aortoiliac occlusive disease. *J Vasc Surg* 1997;**25**:365—79.